

EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	267	GDH and mutant	US-PGPUB; USPAT; DERWENT	OR	ON	2006/06/23 13:12
L2	55833	thr	US-PGPUB; USPAT; DERWENT	OR	ON	2006/06/23 13:12
L3	99	l1 and l2	US-PGPUB; USPAT; DERWENT	OR	ON	2006/06/23 13:12
L4	1	l1 and thr366	US-PGPUB; USPAT; DERWENT	OR	ON	2006/06/23 13:13

Connecting via Winsock to STN

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TERMINAL (ENTER 1, 2, 3, OR ?):2

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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS	4	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS	5	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS	6	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS	7	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS	8	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS	9	MAR 22	EMBASE is now updated on a daily basis
NEWS	10	APR 03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS	11	APR 03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL
NEWS	12	APR 04	STN AnaVist \$500 visualization usage credit offered
NEWS	13	APR 12	LINSPEC, learning database for INSPEC, reloaded and enhanced
NEWS	14	APR 12	Improved structure highlighting in FQHIT and QHIT display in MARPAT
NEWS	15	APR 12	Derwent World Patents Index to be reloaded and enhanced during second quarter; strategies may be affected
NEWS	16	MAY 10	CA/CAPlus enhanced with 1900-1906 U.S. patent records
NEWS	17	MAY 11	KOREAPAT updates resume
NEWS	18	MAY 19	Derwent World Patents Index to be reloaded and enhanced
NEWS	19	MAY 30	IPC 8 Rolled-up Core codes added to CA/CAPlus and USPATFULL/USPAT2
NEWS	20	MAY 30	The F-Term thesaurus is now available in CA/CAPlus
NEWS	21	JUN 02	The first reclassification of IPC codes now complete in INPADOC
NEWS EXPRESS			FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005. V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT http://download.cas.org/express/v8.0-Discover/
NEWS HOURS			STN Operating Hours Plus Help Desk Availability
NEWS LOGIN			Welcome Banner and News Items
NEWS IPC8			For general information regarding STN implementation of IPC 8
NEWS X25			X.25 communication option no longer available after June 2006

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 13:59:34 ON 23 JUN 2006

=> file medline

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

FILE 'MEDLINE' ENTERED AT 14:00:01 ON 23 JUN 2006

FILE LAST UPDATED: 22 JUN 2006 (20060622/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).

See also:

<http://www.nlm.nih.gov/mesh/>

http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s gdh and mutant

1031 GDH

54 GDHS

1045 GDH

(GDH OR GDHS)

169375 MUTANT

98956 MUTANTS

225610 MUTANT

(MUTANT OR MUTANTS)

L1 124 GDH AND MUTANT

=> s l1 and (thr or t)

13371 THR

153 THRS

13439 THR

(THR OR THRS)

4221788 T

L2 103 L1 AND (THR OR T)

=> s l2 and (thr366 or t366)

4 THR366

3 T366

L3 0 L2 AND (THR366 OR T366)

=> l2 and 366

L2 IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.

For a list of commands available to you in the current file, enter

"HELP COMMANDS" at an arrow prompt (=>).

=> s l2 and 366

5754 366

L4 0 L2 AND 366

=> l2 and asp

L2 IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l2 and asp

20176 ASP

265 ASPS

20368 ASP

(ASP OR ASPS)

L5 7 L2 AND ASP

=> d ibib abs l5 1-7

L5 ANSWER 1 OF 7 MEDLINE on STN

ACCESSION NUMBER: 2005158903 MEDLINE

DOCUMENT NUMBER: PubMed ID: 15736973

TITLE: Spontaneous chemical reversion of an active site mutation:
deamidation of an asparagine residue replacing the
catalytic aspartic acid of glutamate dehydrogenase.

AUTHOR: Paradisi Francesca; Dean Jonathan L E; Geoghegan Kieran F;
Engel Paul C

CORPORATE SOURCE: Department of Biochemistry, Conway Institute of
Biomolecular and Biomedical Research, University College
Dublin, Belfield, Dublin 4, Ireland.

SOURCE: Biochemistry, (2005 Mar 8) Vol. 44, No. 9, pp. 3636-43.
Journal code: 0370623. ISSN: 0006-2960.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200505

ENTRY DATE: Entered STN: 29 Mar 2005

Last Updated on STN: 10 May 2005

Entered Medline: 6 May 2005

AB A mutant (D165N) of clostridial glutamate dehydrogenase (GDH) in which the catalytic Asp is replaced by Asn surprisingly showed a residual 2% of wild-type activity when purified after expression in Escherichia coli at 37 degrees C. This low-level activity also displayed Michaelis constants for substrates that were remarkably similar to those of the wild-type enzyme. Expression at 8 degrees C gave a mutant enzyme preparation 1000 times less active than the first preparation, but progressively, over 2 weeks' incubation at 37 degrees C in sealed vials, this enzyme regained 90% of the specific activity of wild type. This suggested that the mutant might undergo spontaneous deamidation. Mass spectrometric analysis of tryptic peptides derived from D165N samples treated in various ways showed (i) that the Asn is in place in D165N GDH freshly prepared at 8 degrees C; (ii) that there is a time-dependent reversion of this Asn to Asp over the 2-week incubation period; (iii) that detectable deamidation of other Asn residues, in Asn-Gly sequences, mainly occurred in sample workup rather than during the 2-week incubation; (iv) that there is no significant deamidation of other randomly chosen Asn residues in this mutant over the same period; and (v) that when the protein is denatured before incubation, no deamidation at Asn-165 is detectable. It appears that this deamidation depends on the residual catalytic machinery of the mutated GDH active site. A literature search indicates that this finding is not unique and that Asn may not be a suitable mutational replacement in the assessment of putative catalytic Asp residues by site-directed mutagenesis.

L5 ANSWER 2 OF 7 MEDLINE on STN
 ACCESSION NUMBER: 2001306691 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 11341912
 TITLE: Contribution of an aspartate residue, D114, in the active site of clostridial glutamate dehydrogenase to the enzyme's unusual pH dependence.
 AUTHOR: Coughlan S; Wang X G; Britton K L; Stillman T J; Rice D W; Chiaraluce R; Consalvi V; Scandurra R; Engel P C
 CORPORATE SOURCE: Department of Biochemistry and Conway institute of Biomolecular and Biomedical Research, University College Dublin, Belfield, Ireland.
 SOURCE: Biochimica et biophysica acta, (2001 Jan 12) Vol. 1544, No. 1-2, pp. 10-7.
 Journal code: 0217513. ISSN: 0006-3002.
 PUB. COUNTRY: Netherlands
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200105
 ENTRY DATE: Entered STN: 4 Jun 2001
 Last Updated on STN: 4 Jun 2001
 Entered Medline: 31 May 2001

AB Glutamate dehydrogenase from Clostridium symbiosum displays unusual kinetic behaviour at high pH when compared with other members of this enzyme family. Structural and sequence comparisons with GDHs from other organisms have indicated that the Asp residue at position 114 in the clostridial enzyme may account for these differences. By replacing this residue by Asn, a mutant protein has been created with altered functional properties at high pH. This mutant protein can be efficiently overexpressed in Escherichia coli, and several criteria, including mobility in non-denaturing electrophoresis, circular dichroism (CD) spectra and initial crystallisation studies, suggest a folding and an assembly comparable to those of the wild-type protein. The D114N mutant enzyme shows a higher optimum pH for activity than the wild-type enzyme, and both CD data and activity measurements show that the distinctive time-dependent reversible conformational inactivation seen at high pH in the wild-type enzyme is abolished in the mutant.

L5 ANSWER 3 OF 7 MEDLINE on STN
 ACCESSION NUMBER: 2000167213 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 10702303
 TITLE: Functions of amino acid residues in the active site of Escherichia coli pyrroloquinoline quinone-containing quinoprotein glucose dehydrogenase.
 AUTHOR: Elias M D; Tanaka M; Izu H; Matsushita K; Adachi O; Yamada M
 CORPORATE SOURCE: Department of Biological Chemistry, Faculty of Agriculture, Yamaguchi University, Yamaguchi 753-8515, Japan.
 SOURCE: The Journal of biological chemistry, (2000 Mar 10) Vol. 275, No. 10, pp. 7321-6.
 Journal code: 2985121R. ISSN: 0021-9258.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200004
 ENTRY DATE: Entered STN: 13 Apr 2000
 Last Updated on STN: 13 Apr 2000
 Entered Medline: 3 Apr 2000

AB Several mutants of quinoprotein glucose dehydrogenase (GDH) in Escherichia coli, located around its cofactor pyrroloquinoline quinone (PQQ), were constructed by site-specific mutagenesis and characterized by enzymatic and kinetic analyses. Of

these, critical mutants were further characterized after purification or by different amino acid substitutions. H262A mutant showed reduced affinities both for glucose and PQQ without significant effect on glucose oxidase activity, indicating that His-262 occurs very close to PQQ and glucose, but is not the electron acceptor from PQQH(2). W404A and W404F showed pronounced reductions of affinity for PQQ, and the latter rather than the former had equivalent glucose oxidase activity to the wild type, suggesting that Trp-404 may be a support for PQQ and important for the positioning of PQQ. D466N, D466E, and K493A showed very low glucose oxidase activities without influence on the affinity for PQQ. Judging from the enzyme activities of D466E and K493A, as well as their absorption spectra of PQQ during glucose oxidation, we conclude that Asp-466 initiates glucose oxidation reaction by abstraction of a proton from glucose and Lys-493 is involved in electron transfer from PQQH(2).

L5 ANSWER 4 OF 7 MEDLINE on STN
 ACCESSION NUMBER: 1998371044 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 9705344
 TITLE: Mutant isolation of the Escherichia coli quinoprotein glucose dehydrogenase and analysis of crucial residues Asp-730 and His-775 for its function.
 AUTHOR: Yamada M; Inbe H; Tanaka M; Sumi K; Matsushita K; Adachi O
 CORPORATE SOURCE: Department of Biological Chemistry, Faculty of Agriculture, Yamaguchi University, Yamaguchi 753-8515, Japan.. yamada@agr.yamaguchi-u.ac.jp
 SOURCE: The Journal of biological chemistry, (1998 Aug 21) Vol. 273, No. 34, pp. 22021-7. Journal code: 2985121R. ISSN: 0021-9258.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199809
 ENTRY DATE: Entered STN: 25 Sep 1998
 Last Updated on STN: 25 Sep 1998
 Entered Medline: 17 Sep 1998

AB Several mutants of quinoprotein glucose dehydrogenase (GDH) in Escherichia coli were obtained and characterized. Of these, significant mutants were further characterized by kinetic analysis after purification or by site-directed mutagenesis to introduce different amino acid substitutions. H775R and H775A showed a pronounced reduction of affinity for a prosthetic group, pyrroloquinoline quinone (PQQ), suggesting that His-775 may directly interact with PQQ. D730N and D730A showed low glucose oxidase activity without influence on the affinity for PQQ, Mg²⁺, or substrate, but D730R showed reduced affinity for PQQ. The spectrum of tryptophan fluorescence revealed that the local structure surrounding PQQ was not changed by D730N mutation. Based on these data, we assume that Asp-730 may occur close to PQQ and function as a proton (and also electron) donor to PQQ or acceptor from PQQH₂. Substitutions of Gly-689, that are located at the end of a unique segment of GDH among homologous quinoprotein dehydrogenases, directed reduction of the affinity for PQQ or GDH activity. Therefore, the unique segment and Asp-730 may play a specific role for GDH, which might be related to the intramolecular electron transfer from PQQ to ubiquinone.

L5 ANSWER 5 OF 7 MEDLINE on STN
 ACCESSION NUMBER: 95194599 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 7888103
 TITLE: Altered GABAergic and glutamatergic transmission in audiogenic seizure-susceptible mice.
 AUTHOR: Cordero M L; Ortiz J G; Santiago G; Negron A; Moreira J A
 CORPORATE SOURCE: Department of Pharmacology, University of Puerto Rico,

School of Medicine, San Juan 00936-5067.
CONTRACT NUMBER: 2 SO6 GMO8224 (NIGMS)
SOURCE: Molecular neurobiology, (1994 Aug-Dec) Vol. 9, No. 1-3, pp. 253-8.
Journal code: 8900963. ISSN: 0893-7648.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 27 Apr 1995
Last Updated on STN: 3 Feb 1997
Entered Medline: 18 Apr 1995

AB The C57BL/10 SPS/sps mouse mutant are audiogenic seizure-susceptible. The enzymatic activities of glutamate decarboxylase (GAD), GABA aminotransferase (GABA-T), alanine aminotransferase (ALA-T), aspartate aminotransferase (ASP-T), and glutamate dehydrogenase (GDH) of whole brain supernatant are significantly reduced in these epileptic mice. GABA uptake is decreased in cortex, midbrain, and pons medulla. Previous studies showed the presence of two sodium-dependent GLU uptake systems in normal (SPS/SP) mice. Glutamate Umax by System 1 is significantly decreased in these mice, whereas the Umax value for System 2 is significantly increased in the epileptic mice.

L5 ANSWER 6 OF 7 MEDLINE on STN
ACCESSION NUMBER: 94311821 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8037659
TITLE: The catalytic role of aspartate in the active site of glutamate dehydrogenase.
AUTHOR: Dean J L; Wang X G; Teller J K; Waugh M L; Britton K L; Baker P J; Stillman T J; Martin S R; Rice D W; Engel P C
CORPORATE SOURCE: Krebs Institute for Biomolecular Research, Department of Molecular Biology and Biotechnology, University of Sheffield, U.K.
SOURCE: The Biochemical journal, (1994 Jul 1) Vol. 301 (Pt 1), pp. 13-6.
Journal code: 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 25 Aug 1994
Last Updated on STN: 25 Aug 1994
Entered Medline: 16 Aug 1994

AB A putative catalytic aspartyl residue, Asp-165, in the active site of clostridial glutamate dehydrogenase has been replaced with serine by site-directed mutagenesis. The mutant enzyme is efficiently overexpressed in Escherichia coli as a soluble protein and can be successfully purified by the dye-ligand chromatographic procedure normally employed for the wild-type enzyme. By several criteria, including circular dichroism spectrum, sulphydryl reactivity with Ellman's reagent, crystallization and mobility in non-denaturing electrophoresis, the enzyme appears to be correctly folded. NAD⁺ protects the D165S mutant against modification by Ellman's reagent, suggesting unimpaired binding of coenzyme. In standard assays the specific activity is decreased 10(3)-fold in the reductive amination reaction and 10(5)-fold for oxidative deamination. Kinetic studies show that apparent Km values for NADH and 2-oxoglutarate are almost unchanged. The large reduction in the reaction rate coincides with a weakening of the affinity for ammonium ion (Km > 300 mM, compared with 60 mM for the wild-type). The data are entirely consistent with the direct involvement of D165 in catalysis rather than in the binding of coenzyme or 2-oxoglutarate.

L5 ANSWER 7 OF 7 MEDLINE on STN
 ACCESSION NUMBER: 90370274 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 2395534
 TITLE: The C57BL/10Bg sps/sps mouse: a mutant with
 absence-like seizures; neurochemical and behavioral
 correlates.
 AUTHOR: Ortiz J G; Negron A E; Garcia M T; Rosado J E; Maldonado C
 S
 CORPORATE SOURCE: Department of Pharmacology, University of Puerto Rico
 School of Medicine, San Juan.
 SOURCE: Neuroscience letters, (1990 Jul 3) Vol. 114, No. 2, pp.
 231-6.
 Journal code: 7600130. ISSN: 0304-3940.
 PUB. COUNTRY: Netherlands
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199010
 ENTRY DATE: Entered STN: 9 Nov 1990
 Last Updated on STN: 6 Feb 1998
 Entered Medline: 11 Oct 1990

AB C57BL/10Bg sps/sps mice display behavioral arrest, similar to generalized
 absence seizures. Compared with the parent strain C57BL/10Bg SPS/SPS, the
 activities of glutamate decarboxylase (GAD, E. C. 2.6.1.15), GABA
 aminotransferase (GABA-T, E. C. 2.6.1.19), aspartate
 aminotransferase (ASP-T, E. C. 2.6.1.1), and
 glutamate dehydrogenase (GDH, E. C. 1.4.1.3) in whole brain
 crude supernatant were significantly reduced in the sps/sps mice. Alanine
 aminotransferase activity (ALA-T, E. C. 2.6.1.2), was not
 altered in any of the strains, and normalization of GAD, GABA-T
 and GDH activities by that of ALA-T, further revealed
 significant differences between the normal strain (SPS/SPS), the
 heterozygotes (SPS/sps), and behavioral arrest (sps/sps) mice. These
 results suggest the possible involvement of GABAergic and glutamatergic
 neurotransmission in the absence-like behavior displayed by sps/sps mice.
 Open field behavior of C57BL/10Bg sps/sps mice is characterized by periods
 of marked inactivity which easily distinguish affected homozygotes, from
 their heterozygotes littermates.

=> s 11 and (thr or threonine)
 13371 THR
 153 THRS
 13439 THR
 (THR OR THRS)
 45169 THREONINE
 316 THREONINES
 45313 THREONINE
 (THREONINE OR THREONINES)
 L6 1 L1 AND (THR OR THREONINE)

=> d ibib abs 16

L6 ANSWER 1 OF 1 MEDLINE on STN
 ACCESSION NUMBER: 2002714401 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 12324473
 TITLE: Substitution of Ser for Arg-443 in the regulatory domain of
 human housekeeping (GLUD1) glutamate dehydrogenase
 virtually abolishes basal activity and markedly alters the
 activation of the enzyme by ADP and L-leucine.
 AUTHOR: Zaganas Ioannis; Spanaki Cleanthe; Karpusas Michael;
 Plaitakis Andreas
 CORPORATE SOURCE: Departments of Neurology and Basic Sciences, University of

Crete, School of Health Sciences, Section of Medicine,
71500 Heraklion, Crete, Greece.
SOURCE: The Journal of biological chemistry, (2002 Nov 29) Vol.
277, No. 48, pp. 46552-8. Electronic Publication:
2002-09-24.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200301
ENTRY DATE: Entered STN: 17 Dec 2002
Last Updated on STN: 9 Jan 2003
Entered Medline: 8 Jan 2003

AB Human glutamate dehydrogenase (GDH) exists in GLUD1
(housekeeping) and in GLUD2-specified (brain-specific) isoforms, which
differ markedly in their basal activity and allosteric regulation. To
determine the structural basis of these functional differences, we
mutagenized the GLUD1 GDH at four residues that differ from
those of the GLUD2 isoenzyme. Functional analyses revealed that
substitution of Ser for Arg-443 (but not substitution of Thr for
Ser-331, Leu for Met-370, or Leu for Met-415) virtually abolished basal
activity and totally abrogated the activation of the enzyme by l-leucine
(1-10 mm) in the absence of other effectors. However, when ADP (0.025-0.1
mm) was present in the reaction mixture, l-leucine (0.3-6.0 mm) activated
the mutant enzyme up to >2,000%. The R443S mutant was
much less sensitive to ADP ($SC(50) = 383.9 \pm 14.6$ microm) than the GLUD1
GDH ($SC(50) = 31.7 \pm 4.2$ microm; $p < 0.001$); however, at 1 mm
ADP the $V(max)$ for the mutant (136.67 micromol min⁻¹ mg⁻¹)
was comparable with that of the GLUD1 GDH (152.95 micromol
min⁻¹ mg⁻¹). Varying the composition and the pH of the reaction
buffer differentially affected the mutant and the wild-type
GDH. Arg-443 lies in the "antenna" structure, in a helix that
undergoes major conformational changes during catalysis and is involved in
intersubunit communication. Its replacement by Ser is sufficient to
impair both the catalytic and the allosteric function of human GDH